

EXHIBIT D

UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY

IN RE: BENICAR (OLMESARTAN) PRODUCTS LIABILITY LITIGATION	MDL No. 2606
THIS DOCUMENT RELATES TO ALL CASES	HON. ROBERT B. KUGLER

RULE 26 EXPERT REPORT OF STEPHEN LAGANA, M.D.
REGARDING GENERAL CAUSATION

I. Background and Qualifications

I am a gastrointestinal and liver pathologist at the Columbia University Medical Center in New York, NY. I graduated medical school from the University Of Pittsburgh School Of Medicine in 2008. I performed a residency in anatomic pathology at Columbia University Medical Center from 2008-2011 (and was Chief Resident from 2010-2011). I performed GI/Liver/Surgical Pathology fellowship from 2011-2012 and was subsequently hired as faculty. One of my prime areas of interest has been inflammatory disorders of the small intestine. My practice includes nine months per year of clinical service, of which six include frequent small intestinal biopsies (approximately 10 per day). I work closely with the Celiac Disease Center at Columbia University, and review approximately 100 cases per year outside of my routine practice, at their request. These cases are typically complicated or diagnostically uncertain, and are frequently presented at interdisciplinary conferences to allow for in-depth discussion of both clinical and pathological considerations. Through these experiences, I have personally reviewed or diagnosed numerous cases of olmesartan associated enteropathy (OAE).

Inflammatory conditions of the small intestine also constitute one of my major research interests. I am familiar with the peer reviewed literature in this field. I have published a number of primary research studies and review articles on such disorders.^{1,2,3,4,5} I have also presented related research endeavors to national and international audiences.^{6,7,8,9} I was the senior author of a review article on OAE. My background and experience is set forth as well in my curriculum vitae, attached as Exhibit 1.

II. Scope of Report

You have asked me to provide my opinions as to whether olmesartan medoxomil, marketed under the trade names Benicar, Benicar HCT, Azor, and Tribenzor (collectively referred to in this report as “olmesartan” or “Benicar”), causes organic changes to the small intestine, including villous atrophy, and the condition now known as OAE, or olmesartan enteropathy. The materials and literature primarily relied on are described herein and in the reliance list attached as Exhibit 2. All opinions are provided to a reasonable degree of medical certainty. I am being compensated for my time at a rate of \$500/hour or \$3000/half day, \$5000/full day in court or in a deposition. I have not previously testified in court or on deposition in any lawsuit.

III. Methodology and Literature

The diagnostic approach I take to such cases is standardized, and is the approach utilized in my clinical practice, in connection with the studies I have authored in the peer-reviewed medical literature, and presentations given at major professional meetings. I start with the review of very basic clinical information (generally limited to the presenting symptom; e.g. diarrhea). I then begin my slide review.

Each slide is first assessed on low power (40X magnification). The first question to answer is tissue orientation. To determine this fact, I aim to identify a tissue fragment (or fragments) in which the luminal surface and muscularis mucosae are both visible, and that these two landmarks are displayed in a generally parallel configuration. When a piece fits this description, the next step is the analysis of the crypts and villi (finger-like projections which increase surface area for nutrient absorption). I look for 4 consecutive crypt and villous units which are well oriented, defined as running perpendicularly to the aforementioned luminal surface and muscularis mucosae. In the normal state, the villous should be approximately four times taller than the crypt is deep. If no villi are present (as in cases of villous atrophy), then the crypts alone can suffice for this purpose. If I cannot identify a region that fulfills the criteria described above, then I cannot assess the presence or absence of villous atrophy and will include a comment to that effect in my reports. If I state that the villi are atrophic/normal/something in between, then this implies that I did identify well-oriented tissue fragments.

When this low-power assessment is complete, a medium (100X magnification) and high power (200-400X magnification) examination is performed. The purpose of the higher power examination is to identify patterns of inflammation (if present) and to exclude the presence of an identifiable pathogen. Inflammatory patterns may be subjective or objective. For instance, neutrophils (a white blood cell which is part of the acute inflammatory response) are always abnormal in the intestinal epithelium. Therefore, if they can be identified with certainty, then that is an objective finding which does not truly lend itself to inter-observer disagreement. Another objective measure is the density of intraepithelial lymphocytes (these are a different

type of white blood cell which are part of chronic inflammation which have migrated into the epithelium). Throughout the small intestine, we have an upper limit of normal (which varies from 20-40 intraepithelial lymphocytes per 100 epithelial cells depending on the location in the small intestine). If one can count more than this (which can be accurately estimated with experience), then there is intraepithelial lymphocytosis and inter-observer disagreements, should they occur, can be resolved by simple counting. Some experts employ further testing (i.e. immunohistochemical stains) to identify intraepithelial lymphocytes, whereas others do not. Either approach is scientifically accepted, and one's preference is dependent on the clinical judgment of the pathologist.^{10,11} Such ancillary tissue testing is generally useful only in borderline cases, and in practice I use it in <1% of my clinical cases.

There are two relatively subjective parameters which I also assess for in intestinal biopsies. One is increased lymphocytes and plasma cells (white blood cells which indicate chronic inflammation) and the other is increased eosinophils (white blood cells which can be part of allergic and parasitic reactions, or be present non-specifically in acute and chronic inflammation). These parameters are subjective because lymphocytes, plasma cells, and eosinophils are all present in normal small intestine and there is no well-defined numerical value which separates normal from abnormal. Having seen thousands of such biopsies, I have a significant frame of reference for determining what is normal and what is not. This is a clinical judgment which is employed in clinical and research settings.

During my medium and high-power review of the inflammatory components of the biopsy, I will also be looking for more unusual patterns of injury (e.g. granulomas, subepithelial fibrosis) and common, but non-specific patterns (erosions/ulcers). I will also determine if the normal numbers of all epithelial cell types are present, including goblet (mucus) cells and Paneth (anti-microbial) cells. These cells are classically diminished in number in an extremely rare entity termed autoimmune enteropathy (AIE), although this observation has also been made in OAE.^{2,12}

The final part of my assessment is a high power examination aimed to exclude identifiable pathogens. This involves examining the biopsy while being mindful of where specific pathogens are found and what they look like. It is very rare to identify pathogens in intestinal biopsies, so the search for them is largely an exercise aimed at not overlooking the rare case.

Having gone through the steps described above, I am left with a pattern which includes all the data points described. Patterns are associated with different disease states, and so only after the pattern is established can a thorough consideration of causation be embarked upon. Although there are many patterns and associated causes, let us review the example of the pattern seen in OAE. The typical pattern is atrophic villi, hyperplastic crypts, and increased lymphocytes, plasma cells, and eosinophils. Other frequent, but not universal, findings include neutrophils, intraepithelial lymphocytosis, and subepithelial fibrosis.²

The celiac literature, which is instructive due to the similarity in its pathological presentation to olmesartan enteropathy, recognizes that in some cases there may be a patchy, less diffuse presentation than what is typical.^{13,14} The literature also describes patients diagnosed with OAE, with normal villi, as well as a range of intestinal abnormalities.¹⁵ In cases without definitive pathology, the diagnosis is based on the judgment of the clinician, relying on evaluation of the medical history and clinical course, especially the response to dechallenge (and rechallenge if deemed necessary).¹⁵ A study which we performed at Columbia University raised the possibility of a less severe clinical and pathologic presentation.⁴ If a biopsy displays the characteristic pattern described above, that brings up a number of diseases which can cause those findings (referred to as a differential diagnosis). The most common disease which causes that pattern is celiac disease (gluten sensitive enteropathy).¹⁶ In the United States, celiac disease affects approximately 1% of the population, making it a highly prevalent condition. Every other disease which causes this pattern is either extremely rare (e.g. autoimmune enteropathy-an immune mediated destruction of the intestine which has been reported in only a few adults globally) or the incidence is not entirely certain (e.g. OAE).¹⁷ The diagnosis of celiac disease is often fairly straightforward, but requires correlation between the histology and laboratory values.¹⁸ If I examine a biopsy, and the pattern fits the description above, I will review the medical record in greater depth. If laboratory values related to celiac disease are not available, I will typically issue a report which describes the findings (e.g. "Duodenal mucosa with total villous atrophy, crypt hyperplasia, chronic lymphoplasmacytic inflammation, and intraepithelial lymphocytosis.") and then include a comment about the differential diagnosis. If I have access to all relevant laboratory values, and they suggest celiac disease, then I can make a definitive statement (such as "these findings are consistent with celiac disease"). When I lack necessary data, I try to guide the next steps in evaluation (e.g. "Such findings are typical in celiac disease. Correlation with serologic testing is suggested. If celiac disease work up is negative, other considerations would include medication reactions (olmesartan, mycophenolate, others), and autoimmune conditions").

This approach allows for a reproducible process which focuses on pattern recognition and the subsequent contextualization of the pattern identified by histologic examination. It also suggests next steps to the gastroenterologist and informs them about the direction of my thinking on the case. In many cases encountered in my clinical practice, a discussion with the gastroenterologist would follow after the additional testing has been performed, but the report is designed to be a standalone document.

Through this approach, I have diagnosed patients with intestinal inflammatory changes, villous atrophy, and related gastrointestinal symptoms such as severe, chronic diarrhea and weight loss, as a result of the use of olmesartan. There is no scientific controversy or dispute that olmesartan causes the intestinal changes and resulting clinical symptoms.

The relationship between olmesartan and organic changes to the intestine, most notably villous atrophy, is well established in the medical literature. The first significant publication

regarding olmesartan enteropathy was published by physicians at the Mayo Clinic.¹⁹ Biopsies were examined for each of the 22 patients described. The protocol included evaluation as follows: “The number of intraepithelial lymphocytes per 100 epithelial cells, degree of villous atrophy graded with the modified Marsh classification, presence of subepithelial collagen, degree of lamina propria inflammation, and presence of acute inflammation were assessed. The presence of aberrant or clonal intraepithelial lymphocytes was investigated by CD3 and CD8 immunostaining and polymerase chain reaction, respectively.” The findings of note were discussed: “In all patients, baseline intestinal biopsies demonstrated villous atrophy with variable degrees of mucosal inflammation (Table 2). Total villous atrophy was observed in 15 patients and partial villous atrophy in 7 patients. A thick band of subepithelial collagen (collagenous sprue) was seen in 7 patients... Active/acute inflammation was observed in 15 patients, and increased intraepithelial lymphocytes were found in 14 patients. Aberrant (or clonal) intraepithelial lymphocytes were not detected among the 12 patients tested.” In addition, “Colonoscopy with random colonic biopsies was performed in 13 patients (59%). Microscopic colitis was found in 5 patients (2 had lymphocytic colitis and 3 had collagenous colitis). Biopsies of the stomach were available in 14 patients (64%). Lymphocytic gastritis was diagnosed in 5 patients and collagenous gastritis in 2 patients. Chronic gastritis was diagnosed in an additional 7 patients (1 had *Helicobacter pylori* infection).” These findings demonstrate the range of findings found in patients with olmesartan enteropathy, most notably partial or total villous atrophy and the frequent involvement of other sites in the gut. Follow up biopsy specimens were evaluated: “At the time of this report, follow-up intestinal biopsies have been performed in 18 patients (82%) after a mean of 242.3 days (range, 54-707 days) from the date of suspension of olmesartan. Histologic recovery of the duodenum was documented in 17 patients (Figure). Focal partial villous atrophy was observed in 1 case (patient 2) on a follow-up duodenal biopsy obtained 54 days after suspension of olmesartan.” The authors’ conclusions in this first significant report of olmesartan enteropathy, which was necessarily conservative in discussing association vs. causality, indicated the likely causal relationship: “Resolution of the presenting symptoms and subsequent histologic improvement after suspension of olmesartan, in the absence of clinical evidence of other diseases associated with enteropathy, suggest that the association is not likely to be due to chance.” Finally, the authors discussed aspects of the differential diagnosis and factors useful in diagnosing olmesartan enteropathy: “Pathologic findings in the duodenal biopsy can mimic celiac disease or collagenous sprue. Clinico-pathologic correlation is advised to confirm the diagnosis of olmesartan-associated enteropathy. Pathologic evidence of involvement of other organs (eg, the stomach and colon) suggests that this disorder may affect the entire gastrointestinal tract. We provide evidence of resolution of inflammation and/or fibrosis in the stomach and duodenum after suspension of olmesartan, implying that these changes are associated with the use of olmesartan. Even though follow-up colonoscopies were not performed in the 5 patients with documented microscopic colitis, clinical remission was achieved in all of these patients, a very unlikely outcome in the presence of persistent inflammation or fibrosis of the colon. Recovery of duodenal mucosa in a relatively short time

(median of 8 months from suspension of olmesartan to follow-up biopsies) is a relevant clinical observation because mucosal recovery in other small bowel disorders, such as celiac disease, may take years to occur despite adherence to a gluten-free diet, especially in older adults.” Finally, the authors discuss findings of small bowel bacterial overgrowth in 12 of the patients studied. They commented on this finding as, “intriguing and consistent with prior observations of association of small bowel bacterial overgrowth and enteropathy in symptomatic patients with celiac disease. The reason for this association is unknown. Thus, although small bowel bacterial overgrowth is a well-recognized cause of chronic diarrhea in the right clinical setting, in this series, the lack of clinical response to oral antibiotics suggests that gastrointestinal symptoms are not explained by the effects of an increased number of bacteria in the small bowel.”

This report by the group from the Mayo Clinic illustrates the importance of application of a reasonable differential diagnosis to determine the cause of a patient’s gastrointestinal symptoms, including the interplay between the clinical presentation and findings from intestinal biopsy. One must evaluate the pathologic findings in the context of the clinical presentation and course in order to exclude potential diagnoses, and to ultimately arrive at the most likely diagnosis for the patient’s condition. The potential competing diagnoses in the context of olmesartan enteropathy must be evaluated, and can be ruled out through factors such as a finding of villous atrophy, partial or total, and successful dechallenge with symptoms improving and resolving upon suspension of olmesartan. More recently, some of the same researchers from the Mayo Clinic, and others, provided a biologically plausible mechanism for olmesartan enteropathy, based in part on evaluation of duodenal biopsies, and concluding that there is causality: “In summary, a small number of patients will develop enteropathy in response to olmesartan medoxomil; this enteropathy is not gluten dependent, and both the stomach and colon of many OAE patients are also affected in addition to the small intestine.”²⁰ Although it is still an area of active scientific investigation; it is likely that olmesartan initiates an immune-mediated process.²¹

Following the Rubio-Tapia publication, there have been numerous case reports published in the literature, strongly supporting causality in olmesartan enteropathy. One illustrative case report discusses a patient with hallmark presentation and course for olmesartan enteropathy.²² The patient’s clinical and histopathological course is described in detail: “A 74-year-old Caucasian gentleman with a history of atrial fibrillation, hypertension, and chronic obstructive pulmonary disease was admitted for evaluation of recurrent diarrhea requiring multiple hospital admissions. His symptoms had started a month ago and had gradually worsened, so that he had to be admitted for severe dehydration and acute renal failure...” The patient had negative stool examination, reported weight loss, and a family history of celiac disease, however celiac serology was negative. “Endoscopic evaluation and biopsies were performed. Duodenum showed focally intense acute and chronic inflammation with variable villous flattening. Although results from CD-specific serologic testing were negative, it was thought that seronegative CD could be a possibility. “ The patient did not improve on a gluten-free diet and was hospitalized

for a third time. At this point, his physicians performed a literature search, and found reference to olmesartan enteropathy, at which point a review of medications indicated he was using olmesartan, which was held, resulting in improvement in the diarrhea. The authors observed: “In retrospect, the transient improvements in symptoms were associated with times during which olmesartan was held because of acute kidney injury with relapse occurring after the drug was resumed following normalization of renal function...Olmesartan-induced enteropathy should be in the differential diagnosis for patients who present with severe unexplained chronic diarrhea and weight loss.” This case report demonstrates the application of the differential diagnosis methodology, interplay of pathology and clinical information, and the importance of dechallenge and rechallenge outcomes, in the exclusion of alternate diagnoses and diagnosis of olmesartan enteropathy. Numerous additional case reports provide similar data. ^{23 24 25 26 27 28 29 30}

In our recent systematic review, we summarize the relevant literature on this subject, which I incorporate by reference. (Reference 2, above). We also note that, “it remains likely that due to low awareness of this condition, patients are still being misclassified as having celiac disease or an inflammatory disorder.” It is also important to mention that although we discuss the few isolated reports of similar clinical and pathological presentations with other ARB’s, and allow for the theoretical possibility that ARB enteropathy may exist, there is no consensus that these are anything more than isolated instances, and there is no published study or article suggesting, much less establishing a class effect. In fact, Marthey et. al. conducted a survey of French gastroenterologists, and discovered 36 cases of OAE, compared to 1 case of enteropathy associated with another ARB.¹⁵ References to ARB enteropathy in the article are made in this context. We discuss the approach to diagnosis of olmesartan enteropathy since as we stated in the paper: “Awareness of the spectrum of clinical and histopathologic changes associated with olmesartan use is of great importance to practicing pathologists, as it will avoid misclassification of patients with other disorders and allow for a very simple but powerful intervention (namely, switching antihypertensive medication).” Our discussion of the *histopathologic differential diagnosis* provides a good introduction to the approach to diagnosing the condition: “there is no cardinal finding which can establish the diagnosis of olmesartan-induced injury based solely on histopathology. On the other hand, if one is aware that this entity exists and obtains the relevant history, then the diagnosis is fairly straightforward in most cases.” Ultimately, we discuss the primary competing clinical entities to be excluded in the differential diagnosis, including celiac disease, tropical sprue, autoimmune enteropathy, inflammatory bowel disease (Crohn’s, ulcerative colitis), and other medications (mycophenolate toxicity). Our conclusion on reaching an ultimate diagnosis highlights the proper approach: “Although we have attempted to provide histopathologic features which may aid in the differential diagnosis, definitive diagnosis requires clinico-pathological correlation, highlighting the importance of effective 2-way communication between pathologists and gastroenterologists.”² This is the approach followed at our institution.

It is important to recognize that there are reports of olmesartan enteropathy in the absence of villous atrophy.¹⁵ In fact, we investigated a cohort of patients seen in our endoscopy suite

complaining not of severe diarrhea, but of abdominal pain. We compared patients taking olmesartan containing medications with age and sex matched controls taking other ARBs. We found that there was a trend towards enteropathic histologic findings in the group taking olmesartan compared to other ARBs. This suggests there may be a spectrum of disease, both clinically and pathologically.⁴

IV. Opinion

It is accepted in the medical community, including in the peer reviewed medical literature, without controversy, that some number of patients develops inflammation, villous atrophy, and other intestinal organic changes, and a spectrum of related gastrointestinal harm and symptoms including for example malabsorption, dehydration, chronic diarrhea, chronic vomiting, severe weight loss, abdominal pain, and nausea, as a result of the use of olmesartan medoxomil. This condition is variously described as olmesartan enteropathy, olmesartan-associated enteropathy, olmesartan-induced enteropathy, and in earlier publications, sprue-like enteropathy. As described above, there are also reports of patients diagnosed with olmesartan enteropathy in the absence of villous atrophy. Based on my review of and familiarity with the peer reviewed medical literature, including articles that I have co-authored, as well as my experience in a clinical and research setting, applying the scientifically accepted methods set forth above, there can be no reasonable dispute that this causality exists.

 (LAGANA MD)

November 30, 2016

Stephen M. Lagana, M.D.

¹ Sung D, Iuga AC, Kato T, Martinez M, Remotti HE, **Lagana SM**. *Crypt Apoptotic Body Counts in Normal Ileal Biopsies Overlap with Graft Versus Host Disease and Acute Cellular Rejection of Small Bowel Allografts*. Hum Pathol. 2016 May 28. pii: S0046-8177(16)30098-3. doi: 10.1016/j.humpath.2016.05.017.

² Burbure, Lebwohl, Green, Arguelles-Grande, Bhagat, **Lagana**. *Olmesartan Associated Sprue-like Enteropathy: a Systematic Review with Emphasis on Histopathology*. Hum Pathol. 2016 Apr;50:127-34. doi: 10.1016/j.humpath.2015.12.001

³ Latorre MD, **Lagana MD**, Freedberg MD, Lebwohl MD, Bhagat MD, Lewis MD, Green MD. *Endoscopic biopsy technique in the diagnosis of celiac disease: one bite or two?* Gastrointest Endosc. 2015 Jan 29. pii: S0016-5107(14)02380-3.

⁴ **Lagana SM**, Braunstein ED, Arguelles-Grande C, Bhagat G, Green PH, Lebwohl B. *Sprue-like histology in patients with abdominal pain taking olmesartan compared with other angiotensin receptor blockers.* J Clin Pathol. 2015 Jan;68(1):29-32. doi: 10.1136/jclinpath-2014-202615.

⁵ *The Duodenal Microbiome in Refractory Celiac Disease, Type I.* **Stephen M. Lagana**, Ian S. Cohn, Mara R. Rubenstein, Benjamin Lebwohl, Peter H.R. Green, Govind Bhagat. 105th Annual Meeting of the United States and Canadian Academy of Pathology, Seattle, WA, USA. 3/15/2016

⁶ *Angiotensin receptor blockers other than olmesartan are not associated with histologic evidence of duodenitis.* **Stephen M. Lagana**, Eric Braunstein, Benjamin Lebwohl, Peter H.R. Green. 103rd Annual Meeting of the United States and Canadian Academy of Pathology, San Diego, CA, USA. 3/4/201

⁷ *Apoptotic Body Counts in Normal Ileal Biopsies Overlap With Acute Cellular Rejection of Small-Bowel Allografts.* Diana Sung, MD; Alina Iuga, MD; Helen Remotti, MD; **Stephen M. Lagana**, MD. Annual meeting of the College of American Pathologists 2015 Annual Meeting (CAP '15). Archives of Pathology & Laboratory Medicine: October 2015, Vol. 139, No. 10, pp. e2-e186

⁸ *Histopathologic Outcomes in Olmesartan Related Enteropathy.* Akash Goel, Benjamin Lebwohl, Christina A. Tennyson, Suzanne K. Lewis, Carolina Arguelles-Grande, Peter H. Green, **Stephen M. Lagana**. Digestive Disease Week 2014, Chicago, IL, USA. 5/4/2014.

⁹ *Endoscopic Biopsy Technique and Biopsy Orientation in the Evaluation of Celiac Disease.* Melissa Latorre MD, Stephen M. Lagana MD, Daniel E. Freedberg MD, Benjamin Lebwohl MD, Govind Bhagat MD, Suzanne K. Lewis MD, Peter H.R. Green MD. Digestive Disease Week 2013, Orlando, FL, USA. 8/19/13.

¹⁰ Mino M, Lauwers GY. *Role of lymphocytic immunophenotyping in the diagnosis of gluten-sensitive enteropathy with preserved villous architecture.* Am J Surg Pathol. 2003 Sep; 27(9):1237-42.

¹¹ Hudacko R, Kathy Zhou X, Yantiss RK. *Immunohistochemical stains for CD3 and CD8 do not improve detection of gluten-sensitive enteropathy in duodenal biopsies.* Mod Pathol. 2013 Sep;26(9):1241-5. doi: 10.1038/modpathol.2013.57. Epub 2013 Apr 5.

¹² Masia R, Peyton S, Lauwers GY, Brown I. *Gastrointestinal biopsy findings of autoimmune enteropathy: a review of 25 cases.* Am J Surg Pathol. 2014 Oct;38(10):1319-29.

¹³ Cammarota G, Martino A, Di Caro S, et al. *High-Resolution magnifying upper endoscopy in a patient with patchy celiac disease.* Dig Dis Sci 2005 Mar;50(3):601-4.

¹⁴ Hopper AD, Cross SS, Sander DS. *Patchy villous atrophy in adult patients with suspected gluten-sensitive enteropathy: is a multiple duodenal biopsy strategy appropriate?* Endoscopy 2008 Mar;40(3):213-24.

¹⁵ Marthey, et al. *Olmesartan-associated enteropathy: results of a national survey.* Aliment Pharmacol Ther 2014; 40:1103-1109.

¹⁶ Bao, F, Green PH, Cellier C. *Celiac disease.* N Engl J Med. 2007 Oct 25;357(17):1731-43.

¹⁷ Masia R, Peyton S, Lauwers GY, Brown I. *Gastrointestinal biopsy findings of autoimmune enteropathy: a review of 25 cases.* Am J Surg Pathol. 2014 Oct;38(10):1319-29.

¹⁸ Bao F, Green PH, and Bhagat G. *An update on celiac disease histopathology and the road ahead.* Arch Pathol Lab Med 2012 Jul;136(7):735-45.

- ¹⁹ Rubio-Tapia, MD., Herman, MD., Ludvigsson, MD, PhD, Kelly, PhD, Mangan, MD, Wu, MD, PhD, Murray, MD. *Severe Spruelike Enteropathy Associated with Olmesartan*. Mayo Clin Proc., August 2012;87(8):732-738.
- ²⁰ Marietta EV, Nadeau AM, Cartee AK, Singh I, Rishi A, Choung RS, Wu TT, Rubio-Tapia A and Murray JA. Immunopathogenesis of olmesartan-associated enteropathy. Aliment Pharmacol Ther. 2015 Dec;42(11):1303-14.
- ²¹ Scialom S, Malamut G, Meresse B, Guegan N, Brousse N, Verkarre V, Derrieux C, Macintyre E, Seksik P, Savoye G, Cadiot G, Vuitton L, Marthey L, Carbonnel F, Cerf-Bensussan N, Cellier C. Gastrointestinal Disorder Associated with Olmesartan Mimics Autoimmune Enteropathy. PLoS One. 2015 Jun 23;10(6):e0125024.
- ²² Rishi A, Garland K. *Unusual Severe Side Effect of a Commonly Used Drug*. J Clin Hypertens (Greenwich). 2016 Apr;18(4):363.
- ²³ Theophile H, David XR, Miremont-Salame G, Haramburu F. *Five cases of sprue-like enteropathy in patients treated by olmesartan*. Digestive and liver disease : official journal of the Italian Society of Gastroenterology and the Italian Association for the Study of the Liver. 2014;46(5):465-469.
- ²⁴ de Fonseca A TA, Moskaluk C. *A case of olmesartan induced enteropathy*. Inflammatory Bowel Disease. 2012;18(S17)
- ²⁵ Ianiro G, Bibbo S, Montalto M, Ricci R, Gasbarrini A, Cammarota G. *Systematic review: sprue-like enteropathy associated with olmesartan*. Alimentary Pharmacology & Therapeutics. 2014;40(1):16-23.
- ²⁶ Abdelghany M, Gonzalez L 3rd, Slater J and Begley C. *Olmesartan associated sprue-like enteropathy and colon perforation*. Case Rep Gastrointest Med. 2014;2014:494098. Epub 2014 Mar 4.
- ²⁷ Gaur V, Albeldawi M and Weber L. *Chronic Diarrhea and Weight Loss*. Gastroenterology. 2014 Feb;146(2):347-591. Epub 2013 Dec 14.
- ²⁸ Dreifuss SE1, Tomizawa Y, Farber NJ, Davison JM, Sohnen AE. *Spruelike enteropathy associated with olmesartan: an unusual case of severe diarrhea*. Case Rep Gastrointest Med. 2013;2013:618071.
- ²⁹ Nielsen JA, Steephen A and Lewin M. *Angiotensin-II inhibitor (olmesartan)-induced collagenous sprue with resolution following discontinuation of drug*. World J Gastroenterol. 2013 Oct 28;19(40):6928-30.
- ³⁰ Stanich PP, Yearsley M and Meyer MM. *Olmesartan-associated sprue-like enteropathy*. J Clin Gastroenterol. 2013 Nov-Dec;47(10):894-5.

STEPHEN M. LAGANA, MD

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LICENSURE

- ♦ **New York State Medical License, unrestricted**
 - January 2011 – present
- ♦ **Board certified in Anatomic Pathology**
 - Diplomate of the American Board of Pathology, granted 7/29/2011

ACADEMIC APPOINTMENTS

- ♦ **Columbia University, New York Presbyterian Hospital, New York, NY**
- ♦ **Assistant Professor** of Pathology with subspecialty expertise in **gastrointestinal and liver** pathology, **Director of Quality Assurance** for Anatomic Pathology, **Co-director of Molecular Testing in Surgical Pathology**
 - July 2012-present
 - Clinical responsibilities include sign out of gastrointestinal, liver, and general surgical pathology cases as well as frozen sections and adult autopsies
 - Gastrointestinal case load includes hospital patients (in-patient and out-patient), outreach cases (from community gastroenterologists), and outside consultations
 - Resident teaching activities include both formal lectures, periodic unknown slide sessions and informal teaching at the microscope
 - Medical student teaching including: lecturer to second year medical students including lectures on non-neoplastic stomach and esophagus, laboratory instructor to first year medical and dental students on normal and abnormal histology
- ♦ **Director of Quality Assurance, Anatomic Pathology**
 - Responsibilities include setting annual QA agenda and submitting goals and reports to Hospital and University
 - Ensure compliance and successful completion of inspections by CAP, NY State, Joint Commission
 - Oversight of each division within Anatomic Pathology, consisting of review of all QA data and periodic inspection of each laboratory in AP
 - Production of annual "report card" for each pathologist tracking turn-around time, case load, frozen section accuracy, peer-review, etc
 - Monitor and adjudicate discrepancies with outside hospitals

- Hospital committee membership:
 - Columbia Oncology Operations Council –ensure department practices comply with published best practices from various non-pathology accrediting agencies (e.g. NCI, American College of Surgery, etc)
 - Assist in inspections by above named groups
 - Columbia Doctors Quality Committee- (Faculty Practice) – ensure that department practices meet and exceed standards of our NY Presbyterian physicians
- Major accomplishments in Quality Assurance
 - Won approval from Graduate Medical Education Committee to claim CME credits for daily Surgical Pathology Consensus/QA Conference, which should result in >1000 CME credit hours being awarded to surgical pathologists with no additional costs to the department (Co-direct course with Director of Surgical Pathology)
 - Increased percentage of randomly peer reviewed cases and re-instituted random peer review of GYN cases
 - Instituted real-time scanning of slides and requisitions for surgical pathology cases to ensure dictations correspond to right patient and right slide
 - Electronic task verification for hospital providers to enable complete recording of report generation and review by initiating provider
- ◆ **Co-director of Molecular Testing in Surgical Pathology**
 - Responsibilities include Interfacing with surgeons, endoscopists, and oncologists to determine optimal standing order algorithm for molecular testing in various tumor types
 - Operationalizing aforementioned algorithms
 - Establishment of ticketing system for one-off requests which are not part of an algorithm
 - Overseeing clerical staff to ensure that tissue arrives at the appropriate laboratory (in-house or reference)
 - Interface with reference labs as needed

EDUCATION

Columbia University Medical Center

New York, NY

Fellow in Surgical and Gastrointestinal Pathology

Completed one-year fellowship combining surgical pathology and gastrointestinal/hepatic pathology July 2011-July 2012

Responsibilities included daily independent sign out of either frozen sections, biopsies, or surgical resections as well as participating in resident teaching, clinical conference presentations and ongoing research projects

Columbia University Medical Center

New York, NY

Anatomic Pathology Residency Program

Completed June 2011

Chief Resident 2010-2011

University of Pittsburgh School of Medicine
Doctor of Medicine, May 2008

Pittsburgh, PA

Hofstra University
Bachelor of Arts, Major Natural Science May 2004
♦ Graduated with Honors

Uniondale, NY

RESEARCH

Peer reviewed publications:

Hemant Varma, Phyllis L. Faust, Alejandro D. Iglesias, **Stephen M. Lagana**, Karen Wou, Michio Hirano, Salvatore DiMauro, Mahesh M. Mansukani, Kirsten E. Hoff, Peter L. Nagy, William C. Copeland, Ali B. Naini. **Whole exome sequencing identifies a homozygous POLG2 missense variant in an infant with fulminant hepatic failure and mitochondrial DNA depletion.** Eur J Med Gen. 2016
<http://dx.doi.org/10.1016/j.ejmg.2016.08.012>

Sung D, Iuga AC, Kato T, Martinez M, Remotti HE, **Lagana SM**. **Crypt Apoptotic Body Counts in Normal Ileal Biopsies Overlap with Graft Versus Host Disease and Acute Cellular Rejection of Small Bowel Allografts.** Hum Pathol. 2016 May 28. pii: S0046-8177(16)30098-3. doi: 10.1016/j.humpath.2016.05.017. [Epub ahead of print] PMID: 27246175

Elizabeth Margolskee; Fei Bao; Anne Koehne de Gonzalez; Roger K Moreira; **Stephen M Lagana**; Anthony N Sireci; Antonia R Sepulveda; Helen E Remotti; Jay H Lefkowitz; Marcela Salomao, MD. **Hepatocellular Adenoma Classification: A Comparative Evaluation of Immunohistochemistry and Targeted Mutational Analysis.** Diagn Pathol. 2016 Mar 9;11(1):27. doi: 10.1186/s13000-016-0475-5.

Nina Burbure, Benjamin Lebwohl, Peter H.R. Green, Carolina Arguelles-Grande, Govind Bhagat, **Stephen M. Lagana**. **Oltmesartan Associated Sprue-like Enteropathy: a Systematic Review with Emphasis on Histopathology.** Hum Pathol. 2016 Apr;50:127-34. doi: 10.1016/j.humpath.2015.12.001.

Taylor SA, Vittorio JM, Martinez M, Fester KA, **Lagana SM**, Lobritto SJ, Ovchinsky N. **Anakina-Induced Acute Liver Failure in an Adolescent Patient with Still's Disease.** Pharmacotherapy. 2016 Jan 9. doi: 10.1002/phar.1677

Lefkowitz J, **Lagana SM**, Kato T. **Hepatocellular Carcinoma in Noncirrhotic Liver with Glycogenotic Foci: Basic Science Meets Genomic Medicine.** Semin Liver Dis. 2015 Nov;35(4):450-6. doi: 10.1055/s-0035-1568986

Stephen Lagana, Susan Hsiao, Fei Bao, Antonia Sepulveda, Roger Moreira, Jay Lefkowitz and Helen Remotti. **HepPar-1 and Arginase-1 Immunohistochemistry in Adenocarcinoma of the Small Intestine and Ampullary Region.** Archives of Pathology & Laboratory Medicine Jun 2015, Vol. 139, No. 6 (June 2015) pp. 791-795

Armando, Del Portillo, **Stephen M, Lagana**, Yuan, Yao, Takeshi, Uehara, Nirag, Jhala, Tapan, Ganguly, Peter, Nagy, Jorge, Gutierrez, Aesis, Luna, Julian, Abrams, Yang, Liu, Randall, Brand, Jorge L, Sepulveda, Gary W, Falk, Antonia R, Sepulveda. **Evaluation of Mutational Testing of Pre-neoplastic Barrett's Mucosa by Next Generation Sequencing of FFPE Endoscopic Samples for Detection of Concurrent Dysplasia and Adenocarcinoma in Barrett's Esophagus.** J Mol Diagn. 2015 Jul;17(4):412-9.

Anne K Koehne de Gonzalez, Marcela A Salomao and **Stephen M Lagana**. **Current concepts in the**

Immunohistochemical evaluation of liver tumors

World J Hepatol 7(10):1403-1411. Published online 2015 June 08. doi: 10.4254/wjh.v7.i10.1403

Lagana SM, Salomao M, Remotti HE, Knisely A.S., Moreira RK. Bile Salt Export Pump: a Sensitive and Specific Immunohistochemical Marker of Hepatocellular Carcinoma. Histopathology. 2015 Mar;66(4):598-602. doi: 10.1111/his.12601.

Melissa Latorre MD, **Stephen M. Lagana MD**, Daniel E. Freedberg MD, Benjamin Lebwohl MD, Govind Bhagat MD, Suzanne K. Lewis MD, Peter H.R. Green MD. **Endoscopic biopsy technique in the diagnosis of celiac disease: one bite or two?** Gastrointest Endosc. 2015 Jan 29. pii: S0016-5107(14)02380-3

Lagana SM, Braunstein ED, Arguelles-Grande C, Bhagat G, Green PH, Lebwohl B. Spurie-like histology in patients with abdominal pain taking olmesartan compared with other angiotensin receptor blockers. J Clin Pathol. 2015 Jan;68(1):29-32. doi: 10.1136/jclinpath-2014-202615.

Hanna RF, Ward TJ, Chow DS, **Lagana SM**, Moreira RK, Emond JC, Weintraub JL, Prince MR. **An evaluation of the sensitivity of MRI at detecting hepatocellular carcinoma in cirrhotic patients utilizing an explant reference standard.** Clin Imaging. 2014 Jun 4. pii: S0899-7071(14)

Hanna RF, Finkelstone LA, Chow DS, Miloushev VZ, Escudero MR, **Lagana SM**, Prince MR. **Nephrogenic systemic fibrosis risk and liver disease.** Int J Nephrol. 2014;2014:679605. doi: 10.1155/2014/679605. Epub 2014 Mar 23. PMID:24778878

Chen, X., **Lagana, SM.**, Poneros, J., Kato, T., Remotti, F., He, H., Kaminsky, D. and Hamele-Bena, D. (2013). **Cytological diagnosis of angiosarcoma arising in an immunosuppressed patient 6 years after multi-visceral transplantation.** Diagn. Cytopathol.. doi: 10.1002/dc.23068

Lagana SM, Moreira RK, Remotti HE, Bao F. Glutamine synthetase, Heat-shock Protein 70 and Glypican-3 in Intra-hepatic Cholangiocarcinoma and Tumors Metastatic to Liver. Appl Immunohistochem Mol Morphol. 2013 May;21(3):254-7. doi: 10.1097/PAI.0b013e3182642c9c.

Lagana SM, Salomao M, Bao F, Moreira RK, Lefkowitz, J, Remotti HE. Utility of an Immunohistochemical Panel Consisting of Glypican-3, Heat Shock Protein-70, and Glutamine Synthetase in the Distinction of Low-grade Hepatocellular Carcinoma from Hepatocellular Adenoma. Appl Immunohistochem Mol Morphol. 2013 Mar;21(2):170-6. doi: 10.1097/PAI.0b013e31825d527f.

Lagana SM, Taub RN, Borczuk AC. Utility of Glucose Transporter 1 in the Distinction of Benign and Malignant Thoracic and Abdominal Mesothelial Lesions. Arch Pathol Lab Med. 2012 Jul;136(7):804-9. doi: 10.5858/arpa.2011-0219-OA.

Guo-Xia Tong, Lorenzo Memeo, Cristina Colarossi, Diane Hamele-Bena, Cristina Magi-Galluzzi, Ming Zhou, **Stephen M. Lagana**, Lara Harik, Jennifer M. Oliver-Krasinski, Mahesh Mansukhani, Lorenzo Falcone, Hanina Hibshoosh, and Kathleen O'Toole. **PAX8 and PAX2 Immunostaining Facilitates the Diagnosis of Primary Epithelial Neoplasms of the Male Genital Tract.** Am J Surg Pathol. 2011 Oct;35(10):1473-83.

Lagana SM, Hanna RF, Borczuk AC. Pleomorphic (Spindle and Squamous Cell) Carcinoma Arising in a Peripheral Mixed Squamous and Glandular Papilloma in a 70 Year Old Man. Arch Pathol Lab Med. 2011 Oct;135(10):1353-6.

Lagana SM, Moreira RK, Lefkowitz JH. Hepatic Granulomas: Pathogenesis and Differential Diagnosis. Clin Liver Dis. 2010 Nov;14(4):605-17. PMID: 21055685

Lagana S, Parwani AV, Nichols L. Cardiac Sarcoidosis. A Pathology Focused Review. Arch Pathol Lab Med. 2010 Jul;134(7):1039-46.

Nichols L, **Lagana S, Parwani A. Coronary Artery Aneurysm: a Review and Hypothesis Regarding Etiology.** Arch Pathol Lab Med. 2008 May;132(5):823-8. Review.

London B, Michalec M, Mehdi H, Zhu X, Kerchner L, Sanyal S, Viswanathan PC, Pfahnl AE, Shang LL, Madhusudanan M, Baty CJ, **Lagana S, Aleong R, Gutmann R, Ackerman MJ, McNamara DM, Weiss R, Dudley SC Jr. Mutation in glycerol-3-phosphate dehydrogenase 1 like gene (GPD1-L) decreases cardiac Na⁺ current and causes inherited arrhythmias.** Circulation. 2007 Nov 13;116(20):2260-8.

Invited editorials:

Stephen M. Lagana, Julian A. Abrams. A phenotypic analysis shows eosinophilic esophagitis is a progressive fibrostenotic disease. Gastrointest Endosc. 2014 Apr;79(4):586-8. doi: 10.1016/j.gie.2013.12.008

Platform presentations/Abstracts:

The Duodenal Microbiome in Refractory Celiac Disease, Type 1. Stephen M. Lagana, Ian S. Cohn, Mara R. Rubenstein, Benjamin Lebwohl, Peter H.R. Green, Govind Bhagat. 105th Annual Meeting of the United States and Canadian Academy of Pathology, Seattle, WA, USA. 3/15/2016

Angiotensin receptor blockers other than olmesartan are not associated with histologic evidence of duodenitis. Stephen M. Lagana, Eric Braunstein, Benjamin Lebwohl, Peter H.R. Green. 103rd Annual Meeting of the United States and Canadian Academy of Pathology, San Diego, CA, USA. 3/4/2014

Mutational analysis by Next Generation Sequencing of preneoplastic intestinal metaplasia in patients with Barrett Esophagus from endoscopic samples. Lagana, Stephen M.; Yao, Yuan; Uehara, Takeshi; Jhala, Nirag; Ganguly, Tapan; Sepulveda, Jorge; Liu, Yang; Brand, Randall; Falk, Gary W. Sepulveda, Antonia R. European Congress of Pathology, 2013, Lisbon, Portugal. 9/1/13.

A comparison of the Immunophenotype of hepatocellular carcinoma and non-lesional hepatocytes when analyzed with next-generation markers. Stephen Lagana, Marcela Salamao, Fei Bao, Roger Moreira, Jay Lefkowitz and Helen Remotti. 100th Annual Meeting of the United States and Canadian Academy of Pathology, San Antonio, TX, USA. 3/1/2011

Poster presentations/Abstracts:

In-Situ Hybridization for Albumin RNA in Pediatric Liver Cancers Compared to Common Immunohistochemical Markers. Anne K. de Koehne de Gonzalez, Ladan Fazlollahi, Amy Coffey, Helen E. Remotti, **Stephen M. Lagana.** 105th Annual Meeting of the United States and Canadian Academy of Pathology, Seattle, WA, USA. 3/15/2016. Poster 225

Usage of Helicobacter pylori Immunohistochemistry Is Not Associated With the Diagnostic Rate of Helicobacter pylori Infection. Jung Hoon Son, Jill Sink, Benjamin Lebwohl, **Stephen M. Lagana.** 104th Annual Meeting of the United States & Canadian Academy of Pathology March 21-27, 2015 Boston, MA.

Histological Features of Colon Allograft in Intestinal Transplant Patients. Alina Iuga MD, Helen Remotti MD, **Stephen M. Lagana MD**, Aesis M. Luna, Armando Del Portillo MD, PhD, Antonia R. Sepulveda MD, PhD. 104th Annual Meeting of the United States & Canadian Academy of Pathology March 21-27, 2015 Boston, MA.

Apoptotic Body Counts in Normal Ileal Biopsies Overlap With Acute Cellular Rejection of Small-Bowel Allografts. Diana Sung, MD; Alina Iuga, MD; Helen Remotti, MD; **Stephen M. Lagana, MD.** Annual meeting of the College of American Pathologists 2015 Annual Meeting (CAP '15). Archives of Pathology & Laboratory Medicine: October 2015, Vol. 139, No. 10, pp. e2-e186

Utility of an Immunohistochemical Panel Consisting of Glypican-3, Heat-Shock Protein-70, and Glutamine Synthetase in the Evaluation of Hepatoblastoma. Amy Coffee, **Stephen M. Lagana**, Helen Remotti. 104th Annual Meeting of the United States & Canadian Academy of Pathology March 21-27, 2015 Boston, MA.

Use of optical coherence tomography (OCT) in the evaluation of gastric dysplasia. Xu M, **Lagana S**, Sethi A. Presented at Digestive Disease Week, Washington DC 2015

Clinical Characteristics, Associated Conditions, and Medication Use in Patients With Lymphocytic Gastritis. Frager S, Lebwohl B, **Lagana SM**, Green PHR. Presented at Digestive Disease Week, Washington DC 2015

Histopathologic Outcomes in Olmesartan Related Enteropathy. Akash Goel, Benjamin Lebwohl, Christina A. Tennyson, Suzanne K. Lewis, Carolina Arguelles-Grande, Peter H. Green, **Stephen M. Lagana.** Digestive Disease Week 2014, Chicago, IL, USA. 5/4/2014.

HepPar-1 and Arginase-1 in small intestinal and ampullary adenocarcinoma. Stephen Lagana M.D., Susan Hsaio, M.D., Fei Bao, M.D., Antonia Sepulveda M.D. Ph.D., Roger Moreira, M.D., Jay Lefkowitz, M.D., and Helen Remotti, M.D. European Congress of Pathology 2013, Lisbon, Portugal. 9/1/13. Poster 046.

Endoscopic Biopsy Technique and Biopsy Orientation in the Evaluation of Celiac Disease. Melissa Latorre MD, **Stephen M. Lagana MD**, Daniel E. Freedberg MD, Benjamin Lebwohl MD, Govind Bhagat MD, Suzanne K. Lewis MD, Peter H.R. Green MD. Digestive Disease Week 2013, Orlando, FL, USA. 8/19/13.

Bile Salt Export Pump (BSEP): A Sensitive and Specific Marker of Hepatocytic Differentiation in Liver Tumors. **Lagana SM**, Remotti H, Moreira RK. 101st Annual meeting of the United States and Canadian Academy of Pathology, Vancouver, BC, Canada. Poster #248. Tuesday, March 20, 2012.

CD146 Expression in Pleural and Peritoneal Mesothelioma. **Lagana SM**, Taub RN, Borczuk AC. 101st Annual meeting of the United States and Canadian Academy of Pathology, Vancouver, BC, Canada. Poster #296. Wednesday, March 21, 2012.

Immunophenotypic Subtypes of Hepatic Adenomas in a Large Tertiary Care Center in the United States. McMillen E, **Lagana SM**, Bao F. 101st Annual meeting of the United States and Canadian Academy of Pathology, Vancouver, BC, Canada. Poster #255. Tuesday, March 20, 2012.

Glutamine synthetase, Heat-shock Protein 70, and Glypican-3 in Intra- hepatic Cholangiocarcinoma. **SM Lagana**, Roger K. Moreira, Helen Remotti, Fei Bao. American Society of Clinical Pathology. Las Vegas, NV. October 21, 2011

Despite Warning, Gadolinium Is Safe in Patients with Liver Disease. R F Hanna, MD, New York, NY; M R Escudero, MD; V Miloushev, MD; **S Lagana; MD**; E Kulon, MD; M R Prince, MD, PhD. Radiological Society of North America, 97th Annual meeting. Chicago, IL. November 27, 2011

MRI has greater sensitivity than 64-slice CT for detecting hepatocellular carcinoma in cirrhotic patients.

Robert F Hanna, **Stephen Lagana**, Roger K Moreira, Inna Postolov, Jean Emond, Martin R Prince. 19th Annual Meeting and Exhibition of the International Society of Magnetic Resonance in Medicine in Montreal, Canada, May 2011.

Diagnostic and Histogenetic Significance of PAX2 and PAX8 Expression in Three Uncommon Tumors of the Male Lower Urogenital Tract . **Stephen M. Lagana, MD**, Guo-Xia Tong, MD, PhD, Lorenzo Memeo, MD, Cristina Colarossi, MD, Mahesh Mansukhani, MD, Hanina Hibshoosh, MD, and Kathleen O'Toole, MD. American Society of Clinical Pathology Annual Meeting, October 2010, San Francisco, CA.

Glucose Transporter-1 Immunostaining Differentiates Intestinal Metaplasia With Dysplasia From Adenocarcinoma in Barrett Esophagus. **Lagana, SM**, Remotti F, Remotti H. College of American Pathologists Annual Meeting. September 2010, Chicago, IL.

Utility of Glucose Transporter 1 in the differentiation of peritoneal and pleural mesothelioma from non-malignant mesothelium.

SM Lagana, RN Taub and AC Borczuk. United States and Canadian Academy of Pathology 99th Annual Meeting. March 23 2010, Washington, D.C.

A Mutation in the Glycerol-3-Phosphate Dehydrogenase 1-Like Gene (GPD1L) Causes Brugada Syndrome. Barry London, MD, PhD, Shamarendra Sanyal, MD, PhD, Michael Michalec, BS, Arnold E. Pfahnl, MD, PhD, Lijuan L. Shang, PhD, Laurie Kerchner, BS, **Stephen Lagana**, BA, Ryan G. Aleong, MD, Haider Mehdi, PhD, Rebecca Gutmann, RN, BSN, Raul Weiss, MD and Samuel C. Dudley, Jr., MD, PhD. Heart Rhythm Society, Boston MA, May 3, 2006.

3D Kinematic Analysis of Antennular Movements of Caribbean Spiny Lobster (Panulirus argus) in Response to Food Odorants.

Stephen M. Lagana, Peter C. Daniel, PhD. Colonial Academic Association annual meeting, Spring 2004

Invited lectures:

Optimal Immunohistochemical Evaluation of Primary Liver Tumors. Liver evening specialty session. United States and Canadian Academy of Pathology- 2017 Annual Meeting. San Antonio, TX

Villous Atrophy Unrelated to Gluten. Intestinal Immune Based Inflammatory Diseases Symposium. March 8, 2013. Columbia University Faculty House. NY, NY.

The Duodenal Microbiome in Refractory Celiac Disease, Type 1. Celiac Disease Center Research Conference. March 1, 2015

Angiotensin receptor blockers other than olmesartan are not associated with histologic evidence of duodenitis. Celiac Disease Center Research Conference. February 1, 2014

PROFESSIONAL MEMBERSHIPS AND ACTIVITIES

- College of American Pathologists 2008-present
 - **Professional Affairs Committee**, Junior Member (January 2010-December 2011). Junior member of national committee tasked with addressing challenges to the specialty of pathology. Specific concerns include ending abusive self-referral practices, finding innovative ways to deliver the most value to patients and clinicians through optimization of pathology reports and information management systems, private sector advocacy, and the role of the pathologist in accountable care organizations (ACO).
 - **Spokesperson**, as a CAP spokesperson, I am tasked with raising awareness of the

profession of pathology while disseminating useful healthcare related information to the public. I have had radio interviews on subjects ranging from colon cancer to Pap tests which have been broadcast on hundreds of radio stations including the Associated Press Radio Network and Armed Forces Radio.

- Roger C. Haggitt Gastrointestinal Pathology Society (GIPS) 2015-present
- American Gastroenterological Association (AGA) 2015-present
- New York Pathological Society 2013-present
 - Member of Planning Committee (selecting topics, inviting guest speakers)
 - Director of social media outreach via @NYPathSociety twitter handle
- United States and Canadian Academy of Pathology 2010-present
- **Peer reviewer-** Histopathology, BMC Gastroenterology, BMC Cancer, Oncotarget, Journal of Clinical Pathology